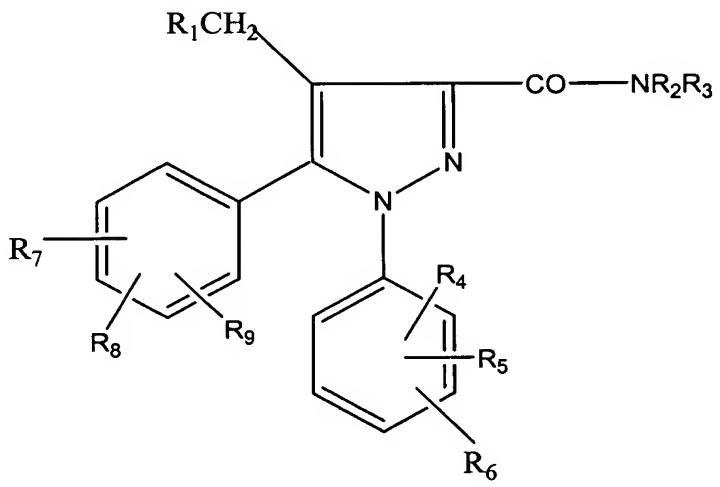
WHAT IS CLAIMED IS:

1. A method of reducing food consumption in a mammal, said method comprising administering to said mammal a first compound which is a PPARα agonist and a second compound which is an antagonist of the CB1 cannabinoid receptor, whereby the consumption of food by the animal is reduced.

2. The method according to claim 1, wherein the PPARα agonist is an OEA-like agonist.

3. The method of claim 1, wherein the PPARα agonist is oleoylethanolamide, palmitoylethanolamide or elaidoylethanolamide.

4. The method of claim 1, wherein the antagonist is a pharmaceutically acceptable salt or solvate of a compound of the formula:



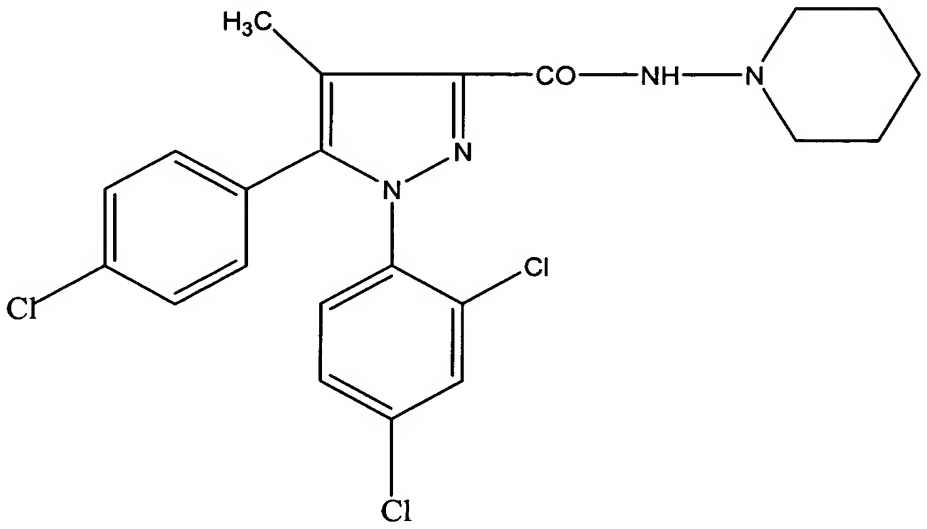
wherein R_1 is hydrogen, a fluorine, a hydroxyl, a (C_1-C_5)alkoxy, a (C_1-C_5)alkylthio, a hydroxy(C_1-C_5)alkoxy, a group -$NR_{10}R_{11}$, a cyano, a (C_1-C_5)alkylsulfonyl or a (C_1-C_5)alkylsulfinyl;

R_2 and R_3 are a (C_1-C_4)alkyl or, together with the nitrogen atom to which they are bonded, form a saturated or unsaturated 5- to 10-membered heterocyclic radical which is unsubstituted or monosubstituted or polysubstituted by a (C_1-C_3)alkyl or by a (C_1-C_3)alkoxy;

R_4, R_5, R_6, R_7, R_8 and R_9 are each independently hydrogen, a halogen or a trifluoromethyl, and if R_1 is a fluorine, R_4, R_5, R_6, R_7, R_8 and/or R_9 can also be a fluoromethyl, with the proviso that at least one of the substituents R_4 or R_7 is other than hydrogen; and

R_{10} and R_{11} are each independently hydrogen or a $(C_1\text{-}C_5)$alkyl, or R_{10} and R_{11}, together with the nitrogen atom to which they are bonded, form a heterocyclic radical selected from pyrrolidin-1-yl, piperidin-1-yl, morpholin-4-yl and piperazin-1-yl, which is unsubstituted or substituted by a $(C_1\text{-}C_4)$alkyl.

5. The method of claim 4, wherein said antagonist is of the formula:

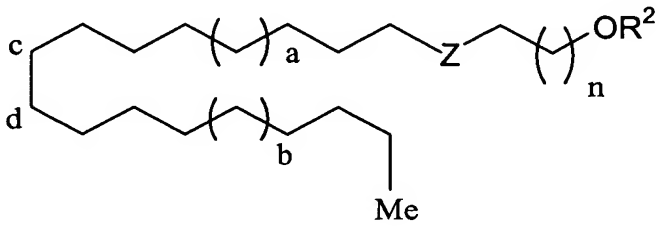


or a pharmaceutically acceptable salt thereof.

6. A method according to claim 1, wherein the mammal is human.

7. A method according to claim 6, wherein said human is overweight or obese.

8. A method according to claim 1, wherein the PPARα agonist is a compound of the following formula:



wherein n is any number from 0 to 5;

the sum of a and b can be any number from 0 to 4;

Z is a member selected from –C(O)N(R^o)-; -(R^o)NC(O)-; -OC(O)-; -(O)CO-; O; NR^o; and S, in which R^o and R^2 are independently selected from the group consisting of substituted or unsubstituted alkyl, hydrogen, substituted or unsubstituted C_1 –C_6 alkyl, substituted or unsubstituted lower (C_1-C_6) acyl, homoalkyl, and aryl;

up to eight hydrogen atoms of the compound may also be substituted by methyl group or a double bond; and

the molecular bond between carbons c and d may be unsaturated or saturated,

or a pharmaceutically acceptable salt thereof.

9. A method according to claim 1, wherein said PPARα agonist is administered with a pharmaceutically acceptable carrier by an oral, rectal, topical, or parenteral route.

10. A method according to claim 1, wherein said antagonist is administered with a pharmaceutically acceptable carrier by an oral, rectal, topical, or parenteral route.

11. A method according to claim 1, wherein said antagonist and said PPARα agonist are administered together.

12. A method according to claim 1, wherein said antagonist and said PPARα agonist are each administered in an amount below their individual ED_{50}.

13. A method according to claim 1, wherein said antagonist and said PPARα agonist are each administered in an amount below their individual ED_{10}.

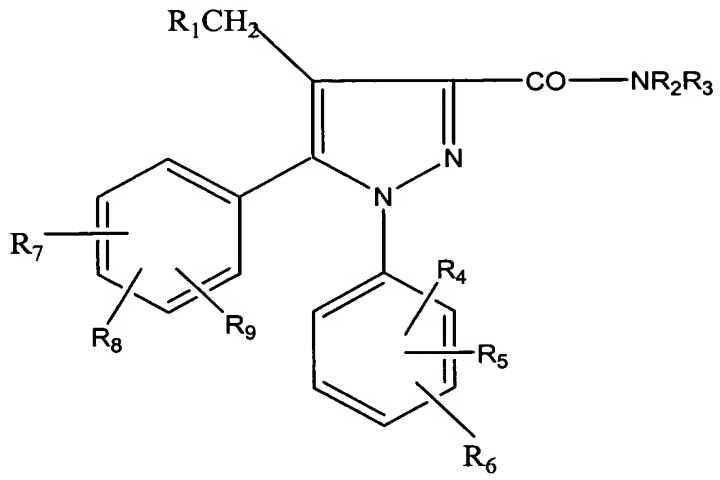
14. A method according to claim 1, wherein at least one of said antagonist and said PPARα agonist is administered in an amount below its ED_{10}.

15. A method according to claim 1, wherein at least one of said antagonist and said PPARα agonist is administered in an amount below its ED_{50}.

16. A pharmaceutical composition for reducing food consumption in a mammal, said composition comprising a PPARα agonist and a cannabinoid CB1 receptor.

17. The composition according to claim 16, wherein the PPARα agonist is oleoylethanolamide.

18. The composition according to claim 17, wherein the antagonist is a pharmaceutically acceptable salt or solvate of a compound of the formula:



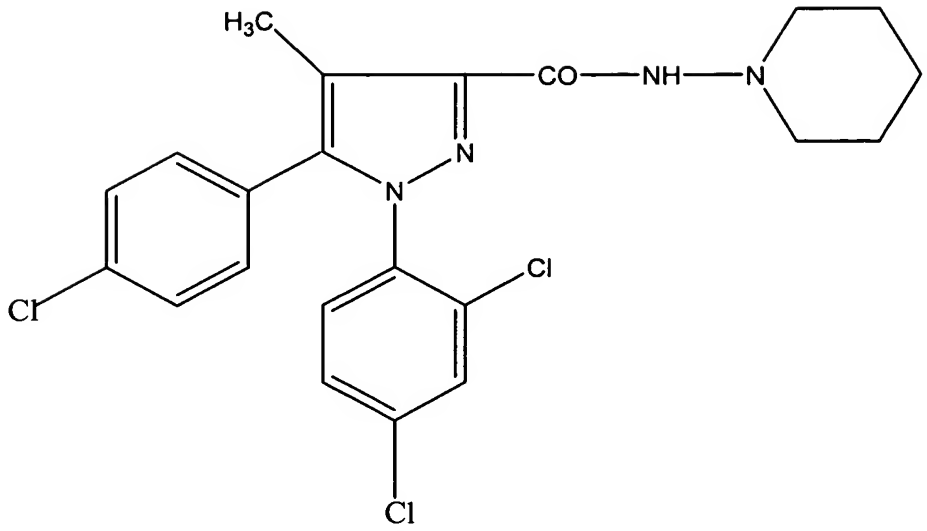
wherein R_1 is hydrogen, a fluorine, a hydroxyl, a (C_1-C_5)alkoxy, a (C_1-C_5)alkylthio, a hydroxy(C_1 -C_5)alkoxy, a group -$NR_{10}R_{11}$, a cyano, a (C_1-C_5)alkylsulfonyl or a (C_1-C_5)alkylsulfinyl;

R_2 and R_3 are a (C_1-C_4)alkyl or, together with the nitrogen atom to which they are bonded, form a saturated or unsaturated 5- to 10-membered heterocyclic radical which is unsubstituted or monosubstituted or polysubstituted by a (C_1-C_3)alkyl or by a (C_1-C_3)alkoxy;

R_4, R_5, R_6, R_7, R_8 and R_9 are each independently hydrogen, a halogen or a trifluoromethyl, and if R_1 is a fluorine, R_4, R_5, R_6, R_7, R_8 and/or R_9 can also be a fluoromethyl, with the proviso that at least one of the substituents R_4 or R_7 is other than hydrogen; and

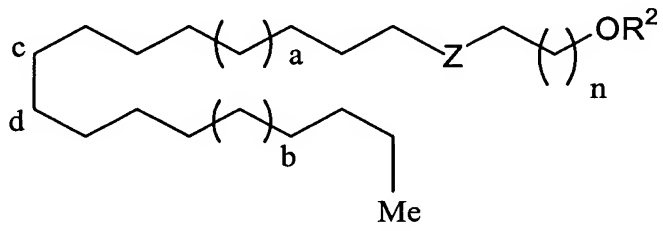
R_{10} and R_{11} are each independently hydrogen or a (C_1-C_5)alkyl, or R_{10} and R_{11}, together with the nitrogen atom to which they are bonded, form a heterocyclic radical selected from pyrrolidin-1-yl, piperidin-1-yl, morpholin-4-yl and piperazin-1-yl, which is unsubstituted or substituted by a (C_1-C_4)alkyl.

19. The composition according to claim 17, wherein said antagonist is of the formula:



or a pharmaceutically acceptable salt thereof.

20. The composition according to claim 17, wherein the PPARα agonist is a fatty acid alkanolamide of the formula:



wherein n is any number from 0 to 5;

the sum of a and b can be any number from 0 to 4;

Z is a member selected from –C(O)N(R^o)-; -(R^o)NC(O)-; -OC(O)-; -(O)CO-; O; NR^o; and S, in which R^o and R^2 are independently selected from the group consisting of substituted or unsubstituted alkyl, hydrogen, substituted or unsubstituted C_1 –C_6 alkyl, substituted or unsubstituted lower (C_1-C_6) acyl, homoalkyl, and aryl;

up to eight hydrogen atoms of the compound may also be substituted by methyl group or a double bond; and

the molecular bond between carbons c and d may be unsaturated or saturated.

21. The composition according to claim 17, wherein said composition is in a formulation suitable for administration by an oral, rectal, topical, or parenteral route of administration.

22. The composition according to claim 17, wherein said composition is in unit dosage format.

23. The composition according to claim 22, wherein at least one of said antagonist and said agonist is in an amount below its ED_{10}.

24. The composition according to claim 22, wherein at least one of said antagonist and said alkanolamide is in an amount below its ED_{50}.

25. The composition according to claim 16, wherein the antagonist has an IC_{50} for the CB1 cannabinoid receptor which is less than one-fourth its IC_{50} for the CB2 cannabinoid receptor.

26. The composition according to claim 20, wherein R^0 and R^2 are members independently selected from the group comprising hydrogen, C_1–C_3 alkyl, and lower (C_1-C_3) acyl.

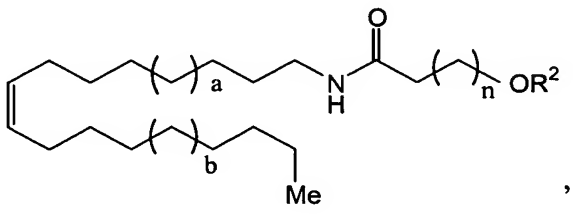
27. The composition according to claim 20, wherein a = 1 and b= 1.

28. The composition according to claim 20, wherein n = 1.

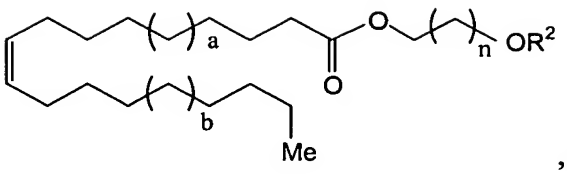
29. The composition according to claim 20, wherein R^1 and R^2 are each H.

30. The composition according to claim 20, wherein the bond between carbon c and carbon d is a double bond.

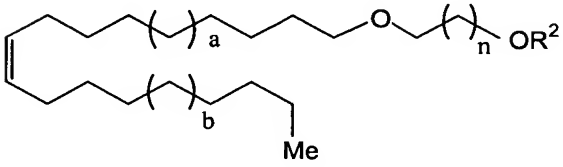
31. The composition according to claim 20, wherein the alkanolamide or its homologue is according to one of the following formulae:



,



, and



,

wherein n is from 1-5 and the sum of a and b is from 0 to 4; R^2 is selected from the group consisting of hydrogen, C_1-C_6 alkyl, and lower (C_1-C_6) acyl; and up to four hydrogen atoms of the fatty acid portion and alkanol portion thereof may also be substituted by methyl or a double bond.

32. A composition of claim 16, wherein the PPARα agonist is selected from the group consisting of clofibrate; fenofibrate, bezafibrate, gemfibrozil, and ciprofibrate.

33. A composition of claim 31, wherein the cannabinoid receptor antagonist is rimonabant.

34. A method of treating an appetency disorder in a human by administering a composition according to claim 17.

35. A method according to claim 34, wherein the appetite for a food, ethanol, or a psychoactive substance is to be reduced.